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A PILOT STUDY ON LEFT VENTRICULAR DIMENSIONS AND WALL STRESS BEFORE AND AFTER SUBMAXIMAL EXERCISE

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ABSTRACT

Left ventricular dimensions and wall stress were measured echocardiographically before and immediately after exercise in 14 athletes and 7 control subjects. Our findings suggest that afterload is an important determinant of cardiac performance and wall hypertrophy in athletes. In spite of major changes in heart rate and blood pressure, left ventricular wall stress remains unchanged following submaximal exercise, in trained and untrained hearts. It would appear that the changes in heart size during exercise are to a large extent limited in untrained ventricles, as smaller left ventricular dimensions are required, to "normalise" wall stress. This results in a lower stroke volume for a given stroke dimensional change. Consequently cardiac output is a function of heart rate rather than stroke volume in untrained subjects. The effect of increased muscle mass in athletes, is to permit larger left ventricular dimensions for a given afterload, thus stroke volume can be augmented. The increase h/R ratio suggests that afterload is more important than preload in the development of left ventricular hypertrophy in rowers and swimmers.

INTRODUCTION

Increased cardiac volume is a feature of athlete's heart. Increased chamber size and/or wall thickness have been documented echocardiographically, the type of athletic endeavour determining whether chamber size or wall thickness is increased. (Gott et al, 1968; Morganroth et al, 1975; Roeske et al, 1976).

The left ventricle hypertrophies in response to a chronic increase in myocardial metabolic rate per beat, the most potent stimulus being increased left ventricular afterload. (Bacleer, 1964). In order to examine the role of increased left ventricular mass in athletes we have studied the relationship between systolic blood pressure, left ventricular chamber size (radius) and systolic wall thickness, before and after submaximal exercise in athletes, and compared the results to untrained control subjects.

METHOD

This is a preliminary report on 21 subjects, seven Olympic rowers (R), seven international swimmers (S), seven untrained students (C). A further 81 subjects have been studied and the results will be published at a later date. Echocardiograms were obtained before and immediately after submaximal upright exercise on a bicycle ergometer. Recordings were obtained after 45 seconds, and both echocardiographic studies were carried out in the same supine position through the same ultrasonic window. Blood pressure and electrocardiogram were recorded simultaneously. Fourteen subjects had two control echocardiograms. The echocardiographic technique used has been described previously (Cahill, 1976).

From the echocardiograms the following standard measurements were made (Figure 1). Left ventricular end diastolic dimension, end systolic dimension, diastolic and systolic wall thickness. Five cardiac cycles were averaged for each parameter. Wall stress was calculated from the equation. (Bennet et al, 1975).

$$\frac{\text{Systolic Blood Pressure}}{\text{Systolic Wall Thickness}} \times \frac{\text{End Systolic Dimension}}{2}$$

In this paper he concludes that there is a close correlation between cuff pressure and left ventricular systolic pressure.

Left ventricular mass and muscle mass was calculated from the echocardiographic measurements by the method of Troy et al, (1972).

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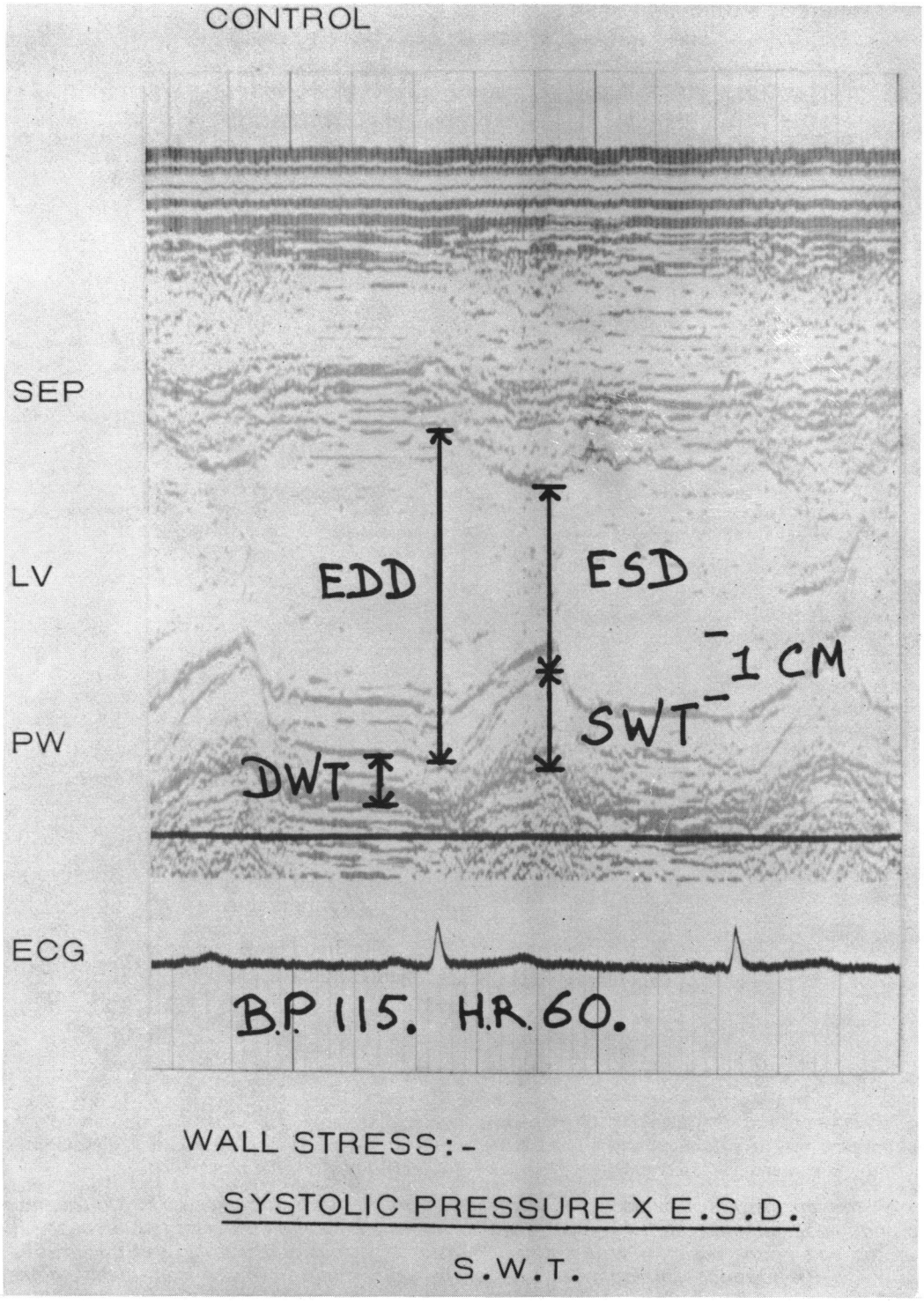

Figure 1: The standard echocardiogram measurements that were made.

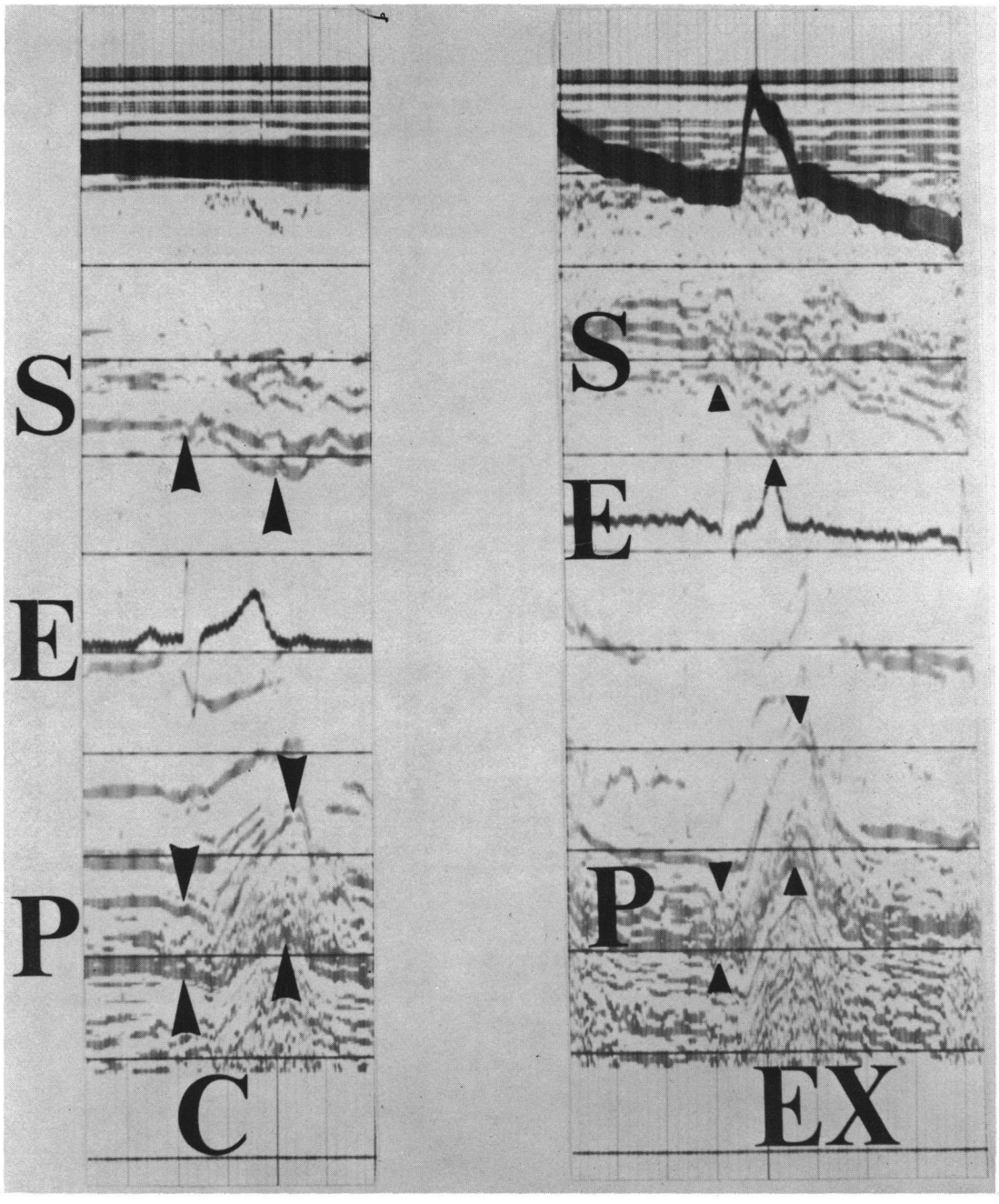

Figure 2 shows the left ventricular endocardial interfaces before (C) and after exercise (EX). The interfaces at end diastole and end systole are indicated by arrows. The dimensions are taken in standard fashion at the R wave of the ECG, and at the end of the posterior wall systolic excursion. Diastolic and systolic wall thickness is also shown. S = septum, E = Ecg, P = posterior wall. Following exercise the end diastolic dimension increase 0.5 cm, the end systolic dimension has decreased 0.8 cm. The systolic wall thickness remains unchanged. Stroke dimensional change has increased from 1.8 cm to 3.0 cm. The velocity of posterior wall movement has increased and ejection time has decreased.

WORK LOAD

The bicycle ergometer was set to a work load of 1200 kiloponds for the controls and swimmers, and 1500 kiloponds for the rowers.

RESULTS

The ages were significantly different between the three groups. Rowers were 27.5 ± 2.4 years, swimmers 18.7 ± 3.2 years. The controls 22.7 ± 3.5 years, were older than the swimmers but younger than the rowers.

The rowers' body surface area was significantly larger 2.1, than either swimmers 1.9, or controls 1.8 m^2.

Athletes had significantly larger left ventricular mass 247 ± 45 grams (R), 232 ± 51 grams (S), than controls 172 ± 33 grams. Only the rowers had significantly greater diastolic posterior wall thickness 1.21 ± 0.19 cm, than either the controls 0.95 ± 0.13 cm, or swimmers 1.1 ± 0.16 cm. There was no significant differ-

TABLE I

Table I shows the values for the three groups of subjects for Age, Body surface area (BSA), left ventricular (LV) Mass, LV muscle mass, and LV diastolic wall thickness.

	CONTROLS	ROWERS	SWIMMERS
Age	22.7 ± 3.5*	27.5 ± 2.4*	18.7 ± 3.2*
BSA	1.8*	2.1*	1.9*
LV mass (grams)	172 ± 33	247 ± 45	232 ± 51
LV Muscle Mass (grams)	45.8	107 (+135%)	77 (+68%)
LV Diastolic Wall Thickness (cm)	0.95 ± 0.13*	1.21 ± 0.19	1.1 ± 0.16
H/R Ratio	.38	.47 (+24%)	.41 (+8%)

* = $P < 0.5$

TABLE II

Table II shows the values for the three groups for heart rate (HR), systolic blood pressure, LV end diastolic dimension, LV end systolic dimension, LV systolic wall thickness, LV wall stress, stroke volume. Cardiac output, and stroke dimensional change, before † and after ** submaximal exercise. * = statistically significant (P 0.05). (Significance levels for stroke dimensional change not available).

	CONTROLS	(P)	ROWERS (R)	(P)	SWIMMERS (S)	(P)
HR	† 74 ± 7	*	58 ± 8		61 ± 9	*
	**117 ± 14	*	84 ± 9		90 ± 26	*
Systolic blood pressure (mm Hg)	† 122 ± 10		117 ± 10		127 ± 14	
	**172 ± 15		186 ± 16		175 ± 25	
LV end diastolic dimension (cm)	† 4.9 ± 0.3		5.1 ± 0.4		5.3 ± 0.4	
	** 4.5 ± 0.5	*	5.4 ± 0.5		5.3 ± 0.4	*
LV end systolic dimension (cm)	† 3.3 ± 0.3		3.4 ± 0.3		3.5 ± 0.3	
	** 2.5 ± 0.5	*	3.2 ± 0.5		3.1 ± 0.3	*
LV systolic wall thickness (cm)	† 1.5 ± 0.1	*	1.9 ± 0.2		1.7 ± 0.2	
	** 1.7 ± 0.3		2.0 ± 0.3		1.8 ± 0.1	
LV wall stress dynes/cm	† 138 ± 21	*	109 ± 17	*	137 ± 26	
	**138 ± 51		151 ± 26		141 ± 27	
Stroke volume	† 89 ± 23		96 ± 24		110 ± 27	
	** 82 ± 26	*	129 ± 35		128 ± 26	*
Cardiac output litres (minute)	† 6.6		5.5		7.0	
	**9.9		10.7		11.0	
Stroke dimensional change	† 1.6		1.6		1.8	
	**2.0		2.2		2.2	

ence between control's and swimmer's diastolic posterior wall thickness. In addition rowers had 135% and swimmers 68% more left ventricular muscle mass than controls. Rowers 107 grams, swimmers 77 grams, controls 49 grams.

Left Ventricular Dimensions:

Athletes had slightly larger end-diastolic dimensions at rest than controls 4.9 ± 0.3 cm, (R) 5.1 ± 0.4 cm, (S) 5.3 ± 0.4 cm. These differences were not significant. After exercise however both groups of athletes had significantly larger end-diastolic dimensions (R) 5.4 ± 0.5 cm, (S) 5.3 ± 0.4 cm, than controls 4.5 ± 0.5 cm. The athletes had increased (R), or maintained their end-diastolic dimension, in contrast to controls who had decreased their end-diastolic dimension.

At rest there was no significant difference between the athlete's and control's end systolic dimension (R) 3.4 ± 0.3 cm (S) 3.5 ± 0.3 cm (C) 3.3 ± 0.3 cm. Following exercise all groups decreased their end systolic dimension, controls to a significantly greater degree than athletes. (C) 2.5 ± 0.5 cm (−24%) (R) 3.2 ± 0.5 cm (−6%) (S) 3.1 ± 0.3 cm (−11%).

Systolic Wall Thickness

The athletes had greater values for systolic wall thickness at rest than controls. This was only significant between controls and rowers. (C) 1.5 ± 0.1 cm, (R) 1.9 ± 0.2 cm, (S) 1.7 ± 0.2 cm. All groups increased their systolic wall thickness after exercise. Controls (1.7 ± 0.3 cm) had a greater increase than athletes (+13%), (R) 2.0 ± 0.3 cm (+5%), (S) 1.8 ± 0.1 cm (+5%), so that systolic wall thickness was not significantly different between the groups after exercise.

Systolic Blood Pressure

There was no significant difference for systolic blood pressure at rest for the three groups. (C) 122 ± 10 mm Hg, (R) 117 ± 10 mm Hg, (S) 127 ± 14 mm Hg. Following exercise the echocardiograms were recorded at similar afterload and no significant difference was observed between any group for systolic blood pressure though the rower's systolic blood pressure was slightly higher (R) 186 ± 16 mm Hg, (C) 172 ± 15 mm Hg, (S) 175 ± 25 mm Hg.

Left Ventricular End Systolic Wall Stress

At rest rowers had significantly lower wall stress 109 ± 17 x 10^3 dyn/cm^2 than the controls 138 ± 21 x 10^3 dyn/cm^2 and swimmers 137 ± 26 x 10^3 dyn/cm^2. This was a function of slightly lower systolic blood pressure and higher systolic wall thickness in rowers. Following exercise there was no significant dif-

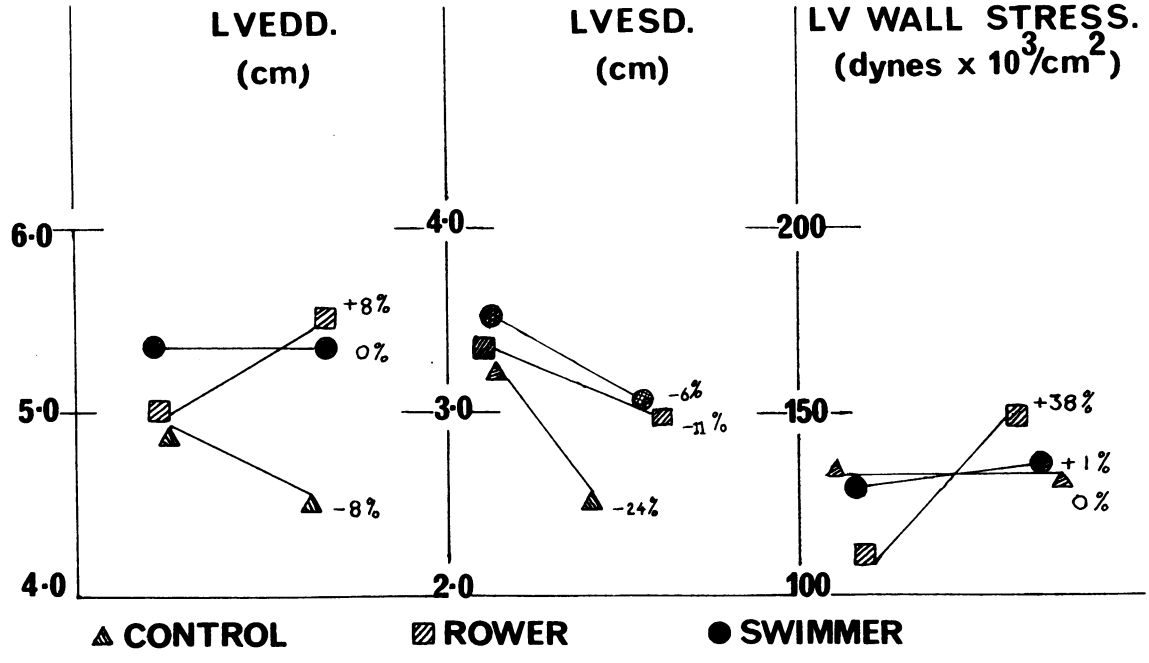

Figure 3 shows the mean and standard deviations before (○) and after (△) exercise for left ventricular end diastolic dimension (LVEDD), left ventricular end systolic dimension (LVESD) and left ventricular wall stress (LV wall stress) for controls (right panel), rowers (middle panel) and swimmers (left panel). See text for discussion. The statistical analysis was a one way analysis of variants.

ference for wall stress between any group (R) $151 \pm 26 \times 10^3$ dyn/cm^2, (S) $141 \pm 27 \times 10^3$ dyn/cm^2, (C) $138 \pm 51 \times 10^3$ dyn/cm^2. Rowers had increased their wall stress by 38% from resting values, but their post exercise wall stress was not significantly different from the other groups. Swimmers and Controls did not change their wall stress significantly.

Heart Rate

As expected the athletes had significantly slower heart rates than controls, both before (C) 74 ± 7, (R) 58 ± 8, (S) 61 ± 9 and after exercise (C) 117 ± 14, (R) 84 ± 9, (S) 90 ± 26. However all groups had a similar significant increase in heart rate after exercise (C) +58%, (R) +44%, (S) +47%. The heart-rates of the athletes returned to resting level much faster than did those of the controls.

Stroke Volume

Athletes had a higher resting stroke volume (R) 96 ± 24cc, (S) 110 ± 27cc than controls 89 ± 23cc. These differences were not significant. Following exercise controls did not increase their stroke volume, 82 ± 26cc in contrast to both groups of athletes who had a significantly higher stroke than controls (R) 129 ± 35cc, (S) 128 ± 26cc.

Stroke Dimension

All groups had a similar stroke dimension at rest (C) 1.6 cm, (R) 1.6 cm, (S) 1.8 cm, and increased their stroke dimension following exercise (C) 2.0 cm (+25%), (R) 2.2 cm (+37%), (S) 2.2 cm (+22%).

Cardiac Output

There was no significant difference for cardiac output between the groups, at rest (C) 6.6 L/min, (R) 5.5 L/min, (S) 7.0 L/min, or after exercise, (C) 9.9 L/min, (R) 10.7 L/min, (S) 11.0 L/min.

DISCUSSION

Echocardiography has been used to evaluate basal and exercise induced changes in left ventricular dimensions, wall thickness and velocity of wall movement. (Kraumz et al, 1970; Redwood et al, 1974; Rost et al, 1975; Redwood et al, 1974). Other techniques available include, serial left ventricular angiograms before and after exercise, or fluorosopy of metal markers sutured to the epicardium at thoracotomy. Harrison et al, (1963). Athletes are not predisposed to catheterization or thoracotomy and therefore the noninvasive techniques used in this study lend themselves to serial studies of left ventricular function in these subjects. The serial reproducability of echocardiography has been documented (Redwood et al, 1974; Troy et al, 1972). We found no significant difference in serial control echocardiographic parameters measured in 14 subjects. All subjects were young and there was no difficulty in identifying rapidly the intracardiac interfaces both before and after exercise. Posture affects cardiac dimensions and stroke volume, (Rushmer 1976). In order to compensate for this all studies were done in the same recumbant position before and after exercise. Echocardiographic parameters were measured in expiration following exercise because of the increased respiratory effort.

Previous investigators have shown that in trained and untrained subjects the left ventricular end systolic dimension decreases with exercise. (Harrison et al, 1963; Gorlin et al, 1965; Stein et al, 1978). The end diastolic volume or dimension has been shown to vary in its response to exercise. Gorlin et al, (1965) found that in 20 subjects, 5 patients increased their end diastolic volume, 7 patients did not change their end diastolic volume, and 8 patients reduced their end diastolic volume during exercise. It is interesting to note that increased stroke volume was only a feature in those patients with increased end diastolic volume. Harrison et al, (1963) noted a decrease in both end diastolic and end systolic dimensions during exercise. Stein et al, (1978) showed that during mild supine exercise there was no change in left ventricular end diastolic dimension. Rushmer found no change in ventricular end diastolic volume, or stroke volume during exercise in conscious dogs in contrast to Harowitz et al, (1968) who noted a decrease in heart size during exercise, as indicated by a more anterior, posterior left ventricular diastolic and systolic wall position.

The role of afterload in determining left ventricular dimensions during exercise has not been evaluated. The systolic pressure elevation in Gorlin's study was mild. Stein et al, did not include serial BP measurement in their study but felt that afterload was not a determinant of left ventricular dimensions and performance. Warner et al, (1964) however showed that the exercise induced elevation of cardiac output can be blocked by prevention of the normal reduction of peripheral resistance with exercise. Braunwald, (1971) outlined four principle determinants of cardiac output, and suggested a "systems analysis" approach may fill in some of the details regarding the interaction of heart rate, afterload, preload and inotropic state. This study is not an attempt to do this. One of the main difficulties is the disparate heart rates between athletes and controls after exercise. Ross et al, (1965) have shown that with moderate supine exercise (10 litres/min/m^2) an increase in cardiac output is achieved by tachycardia, stroke volume remaining unchanged in the majority of subjects. They also showed that tachycardia does not preclude increased stroke volume, during exercise, when heart rate was fixed by atrial pacing prior to exercise. At rest, however, increasing heart rate diminishes stroke volume. Harowitz et al, (1972) showed that stroke volume increased in trained mongrel dogs during strenuous exercise in spite

of a marked increase (+183%) in heart rate. In spite of the significantly different heart rates, all subjects had a similar stroke dimensional change after exercise (C) 0.20 cm, (R) 0.22 cm, (S) 0.22 cm. Thus stroke dimensional change would appear to be independant of heart rate at these exercise levels. Because the control subjects were operating with smaller left ventricular dimensions their stroke volume did not increase, whereas athletes by maintaining or only slightly increasing (R) their end diastolic dimension, produce larger stroke volumes for a similar stroke dimensional change. The issue therefore would appear to be why do control subjects decrease their left ventricular dimensions to such a degree following exercise. Is it a consequence of heart rate, afterload or a combination of both?

The fact that all subjects had similar wall stress both before and after exercise in spite of a considerably increased (+45%) afterload suggests that this factor is a major determinant of left ventricular performance in an *acutely changing situation,* as opposed to a steady state. The drive to increase cardiac output in response to exercise is most efficiently accomplished by increasing both stroke volume and heart rate. As already mentioned for a given stroke dimensional change it is necessary to maintain or increase the left ventricular end diastolic dimension in order to increase stroke volume. If the control subjects had post exercise dimensions of the same order as the athletes then their end systolic wall stress would have increased by 16% and peak systolic wall stress would have increased by 18%. It would appear that small left ventricular dimensions are mandatory in order to maintain wall stress at a constant level and possibly this reflex overwhelms the demand for increased stroke volume.

It would appear reasonable to postulate that the changes in chamber size following exercise are a function of afterload primarily, rather than heart rate and that the faster heart rate produces the increased cardiac output required during exercise when an increase in stroke volume cannot be achieved. Because of the increase in muscle mass found in athletes larger ventricular dimensions and stroke volume can be maintained without increasing wall stress.

The stimulus to hypertrophy in athletes is unclear. It is probably a function of either intermittant pressure and/or volume overload incurred during intensive training. Grossman et al, (1975) have pointed out that the h/R ratio (relationship between diastolic wall thickness h and radius R) reflects the type of stimulus involved in ventricular hypertrophy. Volume overload does not change the h/R ratio, whereas pressure overload increases this ratio. Both groups of athletes had a higher h/R ratio than controls. (C) .38, (R) .47 (+24%), (S) .41 (+8%), suggesting that afterload is a more important determinant of hypertrophy than preload (volume).

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